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PART IV

ENVIRONMENTAL PROTECTION AGENCY

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PESTICIDE PROGRAMS

Rebuttable Presumption Against Registration and Continued Registration of Pesticide Products Containing Maleic Hydrazide

[6560-01]

ENVIRONMENTAL PROTECTION AGENCY

[FRL 607-7; OPP-30000/21]

PESTICIDE PROGRAMS

Rebuttable Presumption Against Registration and Continued Registration of Pesticide Products Containing Maleic Hydrazide

The Deputy Assistant Administrator, Office of Pesticide Programs, Environmental Protection Agency (EPA), has determined that a rebuttable presumption exists against registration and continued registration of all pesticide products containing maleic hydrazide.[1]

I. Regulatory Provisions

A. *General.* Title 40, § 162.11, of the Code of Federal Regulations for the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) as amended (86 Stat. 973, 89 Stat. 751, 7 U.S.C. 136 *et seq.*), provides that a rebuttable presumption against registration shall arise if the Agency determines that a pesticide meets or exceeds any of the risk criteria relating to acute and chronic toxic effects set forth in § 162.11(a)(3). If it is determined that such a rebuttable presumption has arisen, the regulations require that the registrant be notified by certified mail and afforded an opportunity to submit evidence in rebuttal of the presumption. In addition, the Agency has determined such a rebuttable presumption has arisen, the regulations require that the registrant be notified by certified mail and afforded an opportunity to submit evidence in rebuttal of the presumption. In addition, the Agency has determined that the public should also be given notice of the bases for the presumption to provide an opportunity for comment and to solicit additional information relevant to the presumption.

A notice of rebuttable presumption against registration is issued when the evidence related to risk meets or exceeds the criteria set forth in § 162.11(a)(3). It is emphasized that a notice of rebuttable presumption against registration and continued registration of a pesticide is not a notice of intent to cancel the registration of a pesticide, and may or may not lead to cancellation. The notice of intent to cancel is issued only after the risks and benefits of the use(s) of a pesticide are carefully considered and it is determined that the use(s) of the pesticide may generally cause unreasonable adverse effects to the environment.

Accordingly, all registrants and applicants for registration are invited pursuant to 40 CFR 162.11(a)(4) to submit evidence in rebuttal of the presumptions listed in Part II of this notice and, in the case of oncogenicity to submit information which relates to the assessment of oncogenic risks as set forth in the Agency's Interim Procedures and Guidelines for Health Risk and Economic Impact Assessment of Suspected Carcinogens (May 25, 1976; 41 FR 21402). Registrants and other interested parties may also submit for consideration data on benefits which they believe would justify registration or continued registration. In addition, any registrant may petition the Agency to voluntarily cancel a current registration pursuant to Section 6(a)(1) of FIFRA.

B. *Rebuttal Criteria.* Section 162.11(a)(4) provides that a registrant may rebut the presumption by sustaining the burden of proving:

(1) In the case of a pesticide presumed against pursuant to the acute toxicity or lack of emergency treatment criteria, "that when considered with the formulation, packaging, method of use, and proposed restrictions on and directions for use and widesperad and commonly recognized practices of use, the anticipated exposure to an applicator or user and to local, regional, or national populations of nontarget organisms is not likely to result in any significant acute adverse effects" (40 CFR 162.11(a)(4)(i));

(2) In the case of a pesticide presumed against pursuant to the chronic toxicity criteria, "that when considered with proposed restrictions on use and widespread and commonly recognized practices of use, the pesticide will not concentrate, persist or accrue to levels in man or the environment likely to result in any significant chronic adverse effects" (40 CFR 162.11(a)(4)(ii)); or

(3) In either case, that "the determination by the Agency that the pesticide meets or exceeds any of the criteria for risk was in error" (40 CFR 162.11(a)(4)(iii)).

C. *Benefits Information.* In addition to submitting evidence to rebut the presumption of risk, § 162.11(a)(5)(iii) provides that a registrant "may submit evidence as to whether the economic, social and environmental benefits of the use of the pesticide subject to the presumption outweigh the risk of use." If the risk presumptions are not rebutted, the benefit evidence submitted by the registrant,[2] applicants, and other interested persons will be considered by the Administrator in determining the appropriate regulatory action. Specifically, § 162.11(a)(5)(iii) provides that if the "benefits appear to outweigh the risks," the Administrator may issue a notice of intent to hold a hearing pursuant to Section 6(b)(2) of FIFRA rather than a notice of intent to cancel or deny registration pursuant to Section 3(c)(6) of FIFRA. Alternatively, if the "benefits do not appear to outweigh the risks, the Administrator shall issue a notice pursuant to Section 3(c)(6) or Section 6(b)(1) of the Act, as appropriate." Moreover, if at any time the Administrator determines that a pesticide poses an "imminent hazard" to humans or the environment, a notice of suspension may be issued pursuant to Section 6(c) of the Act.

Stated below are the § 162.11(a)(3) risk criteria which the Agency has found to have been met or exceeded by registrations and applications for registration of pesticide products containing maleic hydrazide. The Agency's basis for concluding that these risk criteria have been met or exceeded is set out in "Maleic Hydrazide: Position Document 1," which follows. Copies of attachments to the position Document which are not published with this notice are available for public inspection in the Office of Special Pesticide Reviews. Information protected from disclosure pursuant to FIFRA Section 10 cannot be provided. Specific inquiries concerning the Position Document, as well as requests for access to these files, should be directed to Project Manager Bipin Gandhi, Ph.D., Office of Special Pesticide Reviews (WH-566), EPA, Rm. 447, East Tower, 401 M St. SW., Washington, D.C. 20460 (202-755-8050).

II. Presumptions

A. *Oncogenicity.* 40 CFR 162.11(a)(3)(ii)(A) provides that a rebuttable presumption shall arise if a pesticide "[i]nduces oncogenic effects in experimental mammalian species or in man as a result of oral, inhalation or dermal exposure. . . ." As further clarification of the provision, the preamble to the Agency's Interim Procedures and Guidelines for Health Risk and Economic Impact Assessment of Suspected Carcinogens (May 25, 1976; 41 FR 21402) states that "a substance will be considered a presumptive cancer risk when it causes a statistically significant excess incidence of benign or malignant tumors in humans or animals."

On the basis of scientific studies and information summarized in the Position Document, the Agency has concluded that this risk index has been exceeded by all registrations and applications for

[1] A position document containing an appendix of references, background information, and other material pertinent to the issuance of this notice, has been prepared by the Agency Working Group on maleic hydrazide and is also published with this notice.

[2] Registrants or other interested persons who desire to submit benefit information should consider submitting information on the following subjects, along with any other relevant information they desire to submit:

1. Identification of the major uses of the pesticide, including estimated quantities used by crop or other application.

2. Identification of the minor uses of the pesticide, including estimated quantities used by category such as lawn and garden uses and household uses.

3. Identification of registered alternative products for the uses set forth in (1) and (2) above, including an estimate of their availability.

4. Determination of the change in costs to the user of providing equivalent pesticide treatment with any available substitute products.

5. Assessment of regulation impact upon user productivity (e.g., yield per acre and/or total output) from using available substitute pesticides or from using no other pesticides.

6. If the impacts upon either user costs or productivity are significant, a qualitative assessment of the regulation's impact on production of major agricultural commodities and retail food prices of such commodities.

registration of pesticide products containing maleic hydrazide and that a rebuttable presumption against new or continued registration of such products has therefore arisen.

B. *Mutagenicity.* 40 CFR 162.11(a)(3)(ii)(A) provides that a rebuttable presumption shall arise if a pesticide "(i) nduces mutagenic effects, as determined by multitest evidence."

On the basis of scientific studies and information summarized in the Position Document, the Agency has concluded that this risk index has been exceeded by all registrations and applications for registration of pesticide products containing maleic hydrazide, and that a rebuttable presumption against new or continued registration of such products has therefore arisen.

C. *Other chronic or delayed toxic effects.* 40 CFR 162.11(a)(3)(ii)(B) provides that a rebuttable presumption shall arise of a pesticide "(p) roduces any other chronic or delayed toxic effect in test animals at any dosage up to a level, as determined by the Administrator, which is substantially higher than that to which humans can reasonably be anticipated to be exposed, taking into account ample margins of safety . . . ."

On the basis of scientific studies and information summarized in the Position Document, the Agency has concluded that the risk index for reproductive efforts has been exceeded by all registrations and applications for registration of pesticide products containing diethanolamine salt of maleic hydrazide, and that a rebuttable presumption against new or continued registration of such products has therefore arisen.

III. Grounds for Pesticide Review in Addition to Rebuttable Presumption Criteria

A. *General.* In addition to the risk criteria set forth in 40 CFR 162.11(a)(3) which require the issuance of a notice of rebuttable presumption against registration or continued registration and a determination by the Administrator to register or cancel a pesticide, 40 CFR 162.11(a)(6) provides that the Administrator may determine that a pesticide should be cancelled or that a hearing should be held if the pesticide poses a substantial question of safety to man or the environment "based on toxicological data, epidemiological studies, use history, accident data, monitoring data, or such other evidence as is available to the Administrator."

A determination to cancel or deny registration of a pesticide or to hold a hearing based upon such data and a finding that a pesticide poses a substantial question of safety need not be preceded by public notice and opportunity for rebuttal prior to the administrative adjudicatory hearing procedure of Section 6(b) of the Act. However, where the Agency is using a notice of rebuttable presumption against registration or continued registration based on the risk criteria of 40 CFR 162.11(a)(3), it is in the public interest to include all evidence which may indicate additional grounds for determining that a pesticide causes unreasonable adverse effects on the environment. Accordingly, evidence derived from studies on maleic hydrazide which suggest that N-nitrosodiethanolamine, a metabolite of maleic hydrazide diethanolamine salt for formulations, may produce oncogenic effects and other evidence may form the basis of agency action under the criteria described in 40 CFR 162.11(a)(6). The studies in which these effects have been observed are discussed in the Position Document. Because these effects are of concern to the Agency, it is soliciting relevant information or comment on them.

IV. Information Sought on Reproductive Effects and Translocation of DEA–MH

The information concerning the reproductive effects of diethanolamine salt of maleic hydrazide (DEA–MH) is summarized in the Position Document. DEA–MH caused adverse reproductive effects in test animals at a dose level of 100 mg/kg. However because this level was the only level tested in the study, and there is no information to indicate a no observable effect level or to determine dose-response relationships, it is not possible at this time to estimate with precision the reproductive risks associated with the uses of DEA–MH. Adverse reproductive effects are of concern to the Agency because studies show these effects to be irreversible.

For these reasons, the Agency is soliciting comments on the available evidence. Further, the Agency is requiring that registrants and applicants conduct an additional reproductive effects study and submit the results of the study within one year of the publication of this notice.

DEA residue information currently available was developed using paper chromatography to detect residue levels in potatoes only. Therefore, registrants and applicants having pesticide products containing DEA–MH must submit information of the translocation of DEA–MH to onion bulbs and potato tubers. The Agency recommends that interested registrants and applicants meet with Agency scientists to discuss the adequacy of experimental tests designed to measure reproductive effects and translocation.

V. Registrations and Products Subject to the Notice

All registrants and applicants for registration listed below are being notified by certified mail of the rebuttable presumption existing against registration and continued registration of their products.

The registrants and applicants for registration shall have 45 days from the date this notice is sent or until December 14, 1977, to submit evidence in rebuttal of the presumption. However, the Administrator may, for good cause shown, grant an additional 60 days during which such evidence may be submitted. Notice of such an extension, if granted, will appear in the Federal Register.

A registrant or applicant for registration may, if it desires, assert a business confidentiality claim covering part or all of the information submitted in rebuttal. The registrant or applicant may assert the claim by placing on or attaching to the information a cover sheet, stamped or typed legend, or other suitable form of notice employing language such as "trade secret," "proprietary," or "company confidential." Allegedly confidential portions of otherwise nonconfidential documents should be clearly marked.

If a confidentiality claim is asserted, the information covered by the claim will be disclosed by EPA only to the extent and by means of the procedures set forth in 40 CFR Part 2, Subpart B (41 FR 36906, September 1, 1976). If no confidentiality claim accompanies the information at the time it is received by EPA, EPA will place the information in the public comment file where it will be available for public inspection.

If a registrant or applicant does assert a confidentiality claim for some, but not all, of the information submitted to EPA in rebuttal, the registrant or applicant should furnish two copies of the information to EPA. The first copy should contain all of the information submitted in rebuttal with information claimed as confidential clearly identified. The second copy should be identical to the first except that all information claimed as confidential should be deleted. The second copy will be placed in the public comment file. The first copy will be treated in accordance with the procedures set out above.

VI. Duty To Submit Information on Adverse Effects

Registrants are required by law to submit to EPA any additional information regarding any adverse effects on man or the environment which comes to a registrant's attention at any time, pursuant to Section 6(a)(2) of FIFRA and 40 CFR 162.8(d). If any registrant of maleic hydrazide products has any published or unpublished information, studies, reports, analyses, or reanalyses regarding any adverse effects in animal species or humans, residues, and claimed or verified accidents to humans, domestic animals, or wildlife, which *have not* been previously submitted to EPA, the material must be submitted immediately. When responding to this notice, each registrant shall submit a written certification to the Agency that all information regarding any adverse effects known to the registrant has been submitted. In addition, the registrants should notify EPA of any studies currently in progress, including the purpose of the study, the protocol, the approximate completion date, and a summary of all results observed to date.

VII. Public Comments

During the time allowed for submission of rebuttal evidence, specific comments on the presumptions set forth in this notice and on the material contained in the Position Document are solicited from the public. In particular, any documented episodes of adverse effects

to humans, domestic animals, or wildlife, and information as to any laboratory studies in progress or completed, are requested to be submitted to EPA as soon as possible. Specifically, information on the fate and effects of maleic hydrazide, its impurities, metabolites and degradation products, particularly on animals with metabolism similar to man, is solicited. Similarly, any studies or comments on the benefits from the use of maleic hydrazide are requested to be submitted. All comments and information received, as well as any other relevant information and analysis thereof, which come to the attention of the Agency may serve as a basis for final determination pursuant to § 162.11(a)(5).

All comments and information should be sent to:

Federal Register Section Technical Services Division (WH-569), Rm. 401 East Tower, 401 M Street SW., Washington, D.C. 20460.

Three copies of the comments or information should be submitted to facilitate the work of the Agency and others interested in inspecting them. The comments and information should bear the identifying notation "OPP-30000/21."

Comments and information received within the specified time limit shall be considered before it is determined whether a notice shall be issued in accordance with 40 CFR 162.11(a)(5)(ii).

Comments received after the specified time period will be considered only to the extent feasible, consistent with the time limits imposed by 40 CFR 162.11(a)(5)(ii). All written comments and information filed pursuant to this notice will be available for public inspection in the office of the Federal Register Section from 8:30 a.m. to 4 p.m. during normal working days.

Interested persons are encouraged to take advantage of the opportunity to inspect Agency files during normal working hours since (1) all of the information received may serve as a basis for final determination pursuant to § 162.11(a)(5), and (2) the Agency will not generally publish a summary of information received in the FEDERAL REGISTER at the close of the rebuttal period.

Your cooperation is solicited in identifying any errors or omissions which may have been made in the following computer listings. Corrections to the listings may not necessarily be published in the FEDERAL REGISTER, but rather handled by mail with affected parties. Omissions will be corrected by notice in the FEDERAL REGISTER.

Dated: October 19, 1977.

EDWIN L. JOHNSON,
Deputy Assistant Administrator for Pesticide Programs.

MALEIC HYDRAZIDE: POSITION DOCUMENT 1

MALEIC HYDRAZIDE WORKING GROUP BIPIN GANDHI, PH.D., PROJECT MANAGER, OFFICE OF SPECIAL PESTICIDE REVIEWS, U.S. ENVIRONMENTAL PROTECTION AGENCY

I. BACKGROUND

A. GENERAL CHEMISTRY

Maleic hydrazide or MH [1,2-dihydro-6-pyridazine 3,6-dione (Structure A) or 6-hydroxy-3(2H)-pyridazinone (Structure B)] is a plant-growth regulator in the heterocyclic class of pesticides. MH has an empirical formula of $C_4H_4N_2O_2$ and a molecular weight of 112.09. Its structural formulae are:

Structure A Structure B

MH is slightly acidic and may be titrated as a monobasic acid. It forms salts with diethanolamine (DEA), triethanolamine, and alkalies; but it is stable in acidic and basic solutions. The 6-hydroxy-3 (2H)-pyridazinone isomer can exhibit phenolic properties under physiological conditions.

B. PHYSICAL CHARACTERISTICS

MH is an odorless white crystalline powder. It melts at 296° to 298°C and decomposes above 300°C. It is not decomposed by ultraviolet radiation. MH is soluble in water at concentrations as great as .4g/100 ml at 20°C. MH is also soluble in alkaline solutions. The sodium, potassium, and diethanolamine (DEA) salts are soluble in water and hot alcohol. The solubility of MH in other solvents at 25°C is shown in Table 1.

TABLE 1.—*Solubility of MH*

Solvent:	*Solubility (parts per million)*
Acetone	1,000
Ethyl alcohol	1,000
Dimethyl formamide	20,400
Dimethyl sulfoxide	90,000
Xylene	<1,000

MH has no vapor pressure at room temperature.

C. ENVIRONMENTAL CHARACTERISTICS

The Ecological Monitoring Branch (EMB) of the Technical Services Division within the Office of Pesticide Programs is not currently monitoring for MH in human tissues, agricultural or urban soil, air, or estuarine and ocean organisms (1).

We have seen no evidence that MH persists in water, although it may occur there as a result of runoff. Since MH is not volatile, it is not found in air. Levi and Crafts studied the persistence of MH in heavily treated soils by measuring plant growth in successive harvests (2). The half-life of MH in soil treated with MH in concentrations of 1 to 4 ppm is 40, 7, and 4 days in clay, sand, and muck soils, respectively (3).

The effects of MH on soil bacteria, fungi, and plant pathogens have been extensively researched. In most cases MH slightly inhibits soil bacteria, but it can stimulate soil microflora and fertilize soils. The effects on fungi range from inhibition to stimulation; in some cases MH has no effect. Stimulation occurs indirectly because MH degradation products may be used as a nitrogen source. The fact that no effect is sometimes seen may result from the competition of inhibitory and stimulating effects. Generally, microorganisms in the soil rapidly degrade MH (4). We have seen no reports of other data on environmental chemistry

D. METABOLISM

MH, which is generally translocated from the treated leaves down into the rest of the plant, is relatively stable. Residues occur in potatoes and roots of turf grasses as long as 8 months after treatment (5).

Despite substantial research, the mechanisms of MH metabolism in plants and animals are not well known. Initially, MH was thought to degrade to either maleic or fumeric diamide, which might yield the ammonium salts of the corresponding acids on hydrolysis (6). Towers later concluded that MH in leaf segments of wheat becomes detoxified by the formation of a glycosie (7).

When ^{14}C—MH was applied to specific leafy plants, it migrated from the phloem to the xylem; and mature leaves above the treated leaf became radioactively labelled. Extracts of young wheat seedlings treated with ^{14}C—MH exhibited two raioactive spots on chromatograms and autoradiograms. One spot contained 15 percent of the total radioactivity and was identified and confirmed as the beta-glycoside of MH (7, 8). MH also forms complexes in other plants (9).

There is no clearly documented evidence that hydrazine, a known carcinogen (10), is a metabolite of MH. Biswas and co-workers (11) postulated that when ^{14}C–MH was applied to tea plants it was degraded to lactic acid, succinic acid, maleamide, and hydrazine. However, their data were inadequate to support this conclusion.

Rates of MH absorption vary only moderately among plant species (12). The tomato plant was selected in studies of the effects of plant turgidity, appli-

cation rate, temperature, light, humidity, and formulation on MH absorption. Tests with alkali salts, volatile amines, and nonvolatile amines of MH showed that the nonvolatile amine salts, such as the DEA salt, were absorbed to the greatest extent, followed by the potassium salt to which sorbitol was added in the formulation (12).

Recently, it was reported that tobacco treated with the DEA salt of MH contained as much as 173 ng of N-nitrosodiethanolamine per gram of tobacco, whereas tobacco treated with the potassium salt of MH cotained only 0.1 ng of N-nitrosodiethanolamine per gram of tobacco (13). This finding indicates that DEA may have been the source of N-nitrosodiethanolamine, which is reported to be oncogenic (14).

MH binds in vitro with ribonucleic acid and with proteins such as egg albumin, zein, and hexokinase. Stronger bonds are formed and more MH is bound by the diethanolamine salt of MH than by the potassium salt of MH (9).

When ^{14}C–MH was administered to rats in a single oral dose, they excreted 77 and 12 percent of the compound in urine and feces, respectively, in 6 days. The products excreted in urine were MH (92 to 94 percent) and a conjugate of MH (6 to 98 percent). Only trace amounts of MH (<0.001 percent) were detected in tissues or blood samples after 3 days. Only 0.2 percent of the radioactive MH was excreted through the lungs (15).

El Masri (16) administered single oral doses of MH to rabbits. About 60 percent of the dose was excreted unchanged in the urine. Neither hydrazine, conjugated glucuronic acid, nor ethereal sulfate was excreted.

Barnes and co-workers (17), in a study that substantiates that of El Masri, administered oral doses of MH (100 mg/kg) to rabbits. From 43 to 62 percent of the dose was excreted within 48 hours. The authors did not describe the mode of excretion. Similar results were obtained with a 2-g dose. No glucuronides or ethereal sulfates were detected. The authors speculated that the 40 percent of MH which could not be accounted for may have been more slowly excreted or broken down (17).

E. MONITORING

The Market Basket Survey of the Food and Drug Administration (FDA), which monitors pesticide residues in a sample daily diet of an average 16- to 19-year-old American male in various regions of the country, has not analyzed foods for MH. An FDA official stated in 1974 that "FDA cannot justify surveillance for MH because practical resource constraints, relative risks and the myriad of possible pesticide/food combinations force FDA to make a careful choice of the pesticides and foods which are included in its pesticide program * * *." FDA further stated that it "will seriously consider including MH in the pesticide program if EPA concludes that the toxicity of MH is of such magnitude that periodic testing is needed to protect public health" (18). The MH Working Group knows of no other Federal program to monitor MH.

F. RESIDUES

(1) *Potatoes and onions.* Uniroyal Chemical, one of the primary producers of technical grade MH, has provided EPA with monitoring data on the MH residues in potatoes and onions harvested from 1969 through 1973 (19). The residue data reported for potatoes and onions were within the established tolerance limits (50 ppm and 15 ppm, respectively) when MH–30 (58 percent DEA salt of MH in an inert vehicle) was applied according to label directions (19).

(2) *Tobacco.* Tobacco and cigarettes analyzed for MH residues after the 1970 growing season contained residues of 19 to 31 ppm and 18 to 30 ppm, respectively (20). The Department of Agriculture found average residues of 50 to 100 ppm on tobacco, although in rare cases MH residues exceeded 500 ppm (21).

(3) *Meat, milk, poultry, and eggs.* Dehydrated potato tubers used as feed for beef and dairy cattle, swine, lambs, horses, and poultry can make up 7 to 50 percent of the animal's diet, depending on the animal. If it is assumed that there is no loss of maleic hydrazide in the dehydration process, quantities of MH as low as 15 ppm or as high as 90 ppm could appear in the diets of turkeys or broilers and beef cattle, respectively (12).

The Chemistry Branch of the Registration Division of EPA is not aware of any metabolic studies on large animals or of feeding studies on livestock or poultry (12). Therefore, we are unable to assess the potential for the appearance of secondary residues of MH in meat, milk, poultry, and eggs.

G. PRODUCTION

Section 7(c) of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) requires manufacturers and formulators to submit information on production, distribution, and sales of MH and its formulations to EPA. Under Sections 7(d) and 10 of FIFRA, this information may not be made available to the public. The information is available to the Administrator in a confidential appendix to this report (22).

H. REGISTERED USES AND SUPPLY

(1) *Products.* The diethanolamine salt of MH is registered for use on tobacco, potatoes, onions, and nonbearing citrus trees; it is also registered for use on commercial turf areas that are difficult to mow and not used for grazing, such as grass along highways, airport properties, industrial areas, and golf course roughs. The potassium salt is registered only for use on tobacco.

TABLE 2.—*MH pesticide registrations*

Compound	Federal		Applicants for Federal registrations under 40 CFR 162.17	
	Registrants	Products	Registrants	Products
Maleic hydrazide	5	6	3	3
Diethanolamine salt of MH	42	57	----	----
Potassium salt of MH	5	10	1	1

(2) *Use Patterns.* According to the Government Accounting Office (GAO) report on MH (18), MH formulations are principally used to prevent unwanted suckers on tobacco. It is estimated that about 45 percent of the U.S. tobacco crop acreage is treated with the DEA salt of MH and 45 percent is treated with the potassium salt formulation. Its second greatest use on crops is in the prevention of postharvest sprouting of potatoes and onions. MH is also used to retard grass growth along highways.

Limited data on agricultural applications provided by the EMB (1) show that the use of MH per acre may have declined (Table 3).

	Sites reporting (number)	Sites reporting application (number)	Average total (pounds per acre)
Fiscal year:			
1969	1,684	7	1.43
1970	1,346	3	1.80
1972	1,473	3	3.00
1973	1,402	5	2.25
1974	1,165	2	1.25

(3) *Tolerances.* MH tolerances of 50 ppm in potatoes and 15 ppm in onions (40 CFR 180.175), and a tolerance of 160 ppm for MH as a food additive in potato chips (21 CFR 123.270) have been established. The establishment of tolerances for MH on various foods, particularly potatoes and onions, was necessary because MH is translocated into these foods before harvest. These residues cannot be removed after harvest by washing or any other process.

The World Health Organization (WHO) and the Food and Agricultural Organization (FAO) of the United Nations have not set an acceptable daily intake (ADI) for MH. The ADI is a dosage which, if ingested daily for the lifetime of an individual, is assumed to cause no appreciable risk. No ADI data have been found in EPA files.

I. HUMAN EXPOSURE

Humans are most likely to be exposed to maleic hydrazide in treated potatoes and onions, which are consumed at the rate of 119 and 12.5 pounds per person per year, respectively (23). However, all potatoes and onions may not be treated with MH, and there are no figures available that show the percentage of potatoes and onions that are treated with MH. Users of smoking or chewing tobacco would be further exposed to MH (13).

II. REGULATORY HISTORY

A. *Registration and tolerances.* MH was originally synthesized in 1895, but its ability to regulate plant growth was not discovered until 1949, when Schoene and Hoffman (24) reported that MH has a pronounced, although temporary, inhibitory effect on plant growth, but causes little apparent damage to the plants.

On July 13, 1955, Uniroyal (then the United States Rubber Co.) petitioned FDA to establish tolerances for MH on potatoes, onions, and tobacco of 20, 10, and 10 ppm, respectively. However, this petition was not accepted under Section 408 of the Federal Food, Drug, and Cosmetic Act (FFDCA), because FDA did not think that the use of MH on onions and potatoes to prevent sprouting fit the definition of an economic poison, as defined in FIFRA. Uniroyal requested a public hearing under Section 701(e) of FFDCA to determine if MH was an economic poison. A new petition, submitted October 28, 1956, proposed tolerances of 30, 15, and 75 ppm for potatoes, onions, and potato chips, respectively; it was denied because Uniroyal failed to show that MH was required in the production of potatoes and onions. Tobacco was not included, because it is not regulated under FFDCA. In 1960, in response to another petition submitted April 30, 1959, tolerances of 50, 15, and 160 ppm on potatoes, onions, and potato chips, respectively, were established under the amended FFDCA.

A petition submitted December 10, 1960, by Uniroyal to establish MH tolerances on lemons, peaches, and cranberries of 5, 15, and 20 ppm, respectively, and a petition filed on June 10, 1962, requesting a tolerance of 20 ppm on cranberries were withdrawn because Uniroyal could not provide enough data to support the petitions.

The products containing sodium salt of MH were registered from 1952 through 1967. The registrant, Uniroyal Chemical, did not apply for reregistration. The first registrations of the products containing the DEA and the potassium salts of MH were issued in 1952 and 1969 respectively.

B. INVESTIGATIONS OF ADVERSE EFFECTS

The suspicion that MH may have adverse effects on animals first arose in 1951 when it was claimed that MH is toxic to and retards the growth of frog, toad, and salamander larvae (25).

Between 1951 and 1969 several studies appeared relating MH to oncogenicity, mutagencity, and productive effects. These studies were closely watched by the Agriculture Research Service (ARS), USDA. A major source of concern was a study by Dickens and Jones (26) linking MH to oncogenicity. In response to Dickens and Jones' study, meetings were held October 6 and 7, 1965, to discuss whether MH is oncogenic. The scientists present concluded that MH was only suspect and that more studies with larger numbers and different species of animals were needed (27). Furthermore, both oil and aqueous solutions of MH were used in the studies by Dickens and Jones, and they did not use a control group. In an FDA conference on MH held on October 14, 1965, it was decided that the results reported by Dickens and Jones had no relevance to the safety of MH as a food additive (28).

Although Epstein's research (29) showing that tumors were induced in an area other than the site of injection indicated that MH is carcinogenic, USDA took no regulatory action (30) because FDA did not consider the route of administration in these studies appropriate (31). However, the Director of ARS later requested cancellation of the registration of MH-30 (DEA-MH formulation) because another pesticide had been cancelled on the basis of results of studies in which it was administered parenterally (32). However, no regulatory actions were taken on registrations of products containing MH.

On July 13, 1973, The Honorable Julia Butler Hansen, House of Representatives, requested GAO to provide her with information on Federal testing of MH. The GAO report on MH, published October 23, 1974, reviewed the published scientific findings related to the adverse effects and described three hazards believed to be associated with MH: carcinogenicity, mutagenicity, and reproductive effects (18). GAO recommended that additiona ltesting and research be carried out on MH to determine if it adversely affects humans or the environment. GAO also recommended that FDA periodically test potatoes and onions to make sure that the residues are within the established tolerances (18). Since publication of the GAO report, several more studies of the possible adverse effects of MH on animals have been published.

The 1969 HEW Mrak Report listed MH as a possible oncogenic agent but did not conclude that it was oncogenic (33). MH was among the 120 compounds tested earlier for tumorogenicity by the National Cancer Institute, but it was not found to be carcinogenic (34). More recently, MH was listed as a suspected carcinogen by the Department of Health, Education, and Welfare (35).

C. PESTICIDE EPISODE RESPONSE BRANCH REPORT

Sixteen episodes involving MH have been reported to EPA, two of which were solely related to MH. In the other episodes MH had been used in combination with other pesticides. The Pesticide Episode Response Branch, EPA, stated that there is no significant history of accidents associated with MH and that it appears inappropriate to identify MH as the active agent in these poisonings (36).

D. REFERRAL TO THE OFFICE OF SPECIAL PESTICIDE REVIEWS

MH was referred to OSPR because data indicated that MH may be linked to oncogenic, mutagenic, and reproductive effects.

III. SUMMARY OF SCIENTIFIC EVIDENCE TO SUPPORT A REBUTTABLE PRESUMPTION: CHRONIC TOXICITY

A. ONCOGENIC EFFECTS IN TEST ANIMALS

40 CFR 162.11(a)(3)(ii)(A) provides that "a rebuttable presumption shall arise if a pesticide's ingredient(s) * * * induces oncogenic effects in experimental mammalian species or in man as a result of oral, inhalation, or dermal exposure * * *" Section 162.3(bb) defines the term oncogenic as "the property of a substance or a mixture of substances to produce or induce benign or malignant tumor formation in living animals."

There are a number of studies on MH's oncogenic properties cited in the literature. MH, either as the sodium or potassium salt or as the DEA salt has been applied to rats or mice by injection and by dermal and oral administration. This Agency's Carcinogen Assessment Group (CAG) has evaluated these studies and concluded that MH possesses oncogenic potential. The Working Group concurs with CAG's position. CAG's evaluation of the carcinogenicity of Maleic Hydrazide (37) is summarized below.

(1) *Mouse studies.* Epstein and coworkers (29) found evidence of excess tumor induction in male but not female mice. Their study showed a statistically significant increase of liver tumors (hepatomas) in males injected subcutaneously with 0.1, 0.1, 0.2, and 0.2 ml containing 5, 10, 20 and 20 mg of MH suspended in tricaprylin 1, 7, 14, and 21 days after birth, respectively. After 49 weeks, 4 of the 48 survivors in the control group had hepatic tumors compared to 17 of the 26 survivors in the experimental group.

In a second study, Barnes and coworkers (17) found a statistically significant increase in the total number of tumors, including benign tumors, in female mice fed 1 percent sodium salt of MH (Na-MH) in the diet for 100 weeks. The incidence of tumors in the experimental population (7/8) was significantly higher than that in the control group (0/4) at a p-level equal to 0.01. Tumor incidences in control and experimental male populations were not statistically different.

In the same study, the number of skin tumors in mice to which MH and croton oil were simultaneously applied was not greater than that in mice treated with only croton oil. Furthermore, implantation of cholesterol pellets containing 20 percent MH in the bladders of mice did not cause any increase in the incidence

of tumors in the mice that survived treatment (17).

Innes and co-workers (34) carried out feeding studies on mice for approximately 18 months. Upon completion of these studies, the authors concluded that MH did not cause a significant increase in tumors.

Mukhorina (38) injected one group of mice with the DEA salt of MH (DEA–MH) and fed a second group a diet containing DEA–MH. In neither group was the incidence of tumors significantly above that in control groups.

(2) *Rat Studies.* Six 100-g rats were injected subcutaneously with 2 mg of MH per rat twice weekly for 65 weeks (26). Transplantable sarcomas developed at the site of injection in three of the six experimental animals. None of the six control rats had tumors. As judged by the authors, the study demonstrated that MH is oncogenic; however, the p-value of 0.091 is not significant.

In studies by Barnes and coworkers (17), who administered MH orally and by injection and studies by Hunter and coworkers (39), in which MH and DEA–MH salt were administered by injection, MH did not cause a statistically significant increase in tumor incidence. Data from other studies (40, 41, 42) were found to be inadequate (37, 43), and no conclusions should be drawn from them.

(3) *Conclusion.* The Working Group has concluded from the studies described above that MH is oncogenic in mice. Consequently, the Working Group recommends that a rebuttable presumption be issued on pesticides containing MH pursuant to Section 162.11(a)(3)(ii)(A).

B. MUTAGENIC EFFECTS

40 CFR 162.11(a)(3)(ii)(A) provides that a rebuttable presumption shall arise if a pesticide's ingredient(s), metabolite(s), or degradation product(s) induce mutagenic effects, as determined by multitest evidence. Section 162.3(y) defines the term "mutagenic" as "the property of a substance or mixture of substances to induce changes in the genetic complements of either somatic or germinal tissue in subsequent generations."

This review of the studies is divided into three sections: chromosomal mutations, gene mutations, and other tests bearing on mutagenicity. These studies were evaluated by the Health Effects Research Laboratories (HERL) (44).

(1) *Chromosomal Mutation.* Maleic hydrazide breaks chromosomes of a number of plant species, producing deletions, duplications, and chromosomal rearrangements. The effect is dependent on the stage of the cell cycle, and occurs during the G_1 through S phases (45).

Manna and Parida (46) examined meiotic chromosomes from several grasshopper species which were given MH in food. The mortality rate 48 hours after feeding ranged from 17 to 58 percent, depending on the species; none of the controls died. In treated males chromatid and chromosome breaks were noted at various stages of meiosis; chromosome bridges and lagging chromosomes were seen in anaphase I and II figures; and chromosome stickiness was prevalent in all meiotic stages examined. In control males, no breaks were found, and chromosome stickiness was much less than in treated animals.

Manna and co-workers (47) injected mice with MH (167 mg/kg body weight)[3] and then examined their bone marrow for chromosome aberrations 10 to 120 hours after treatment. The frequency of gaps, chromatid breaks, and translocations significantly increased in treated animals. An increase in isochromatid breaks was not noted, a fact which may indicate that MH does not affect cells in the G_1 phase of the cell cycle. Another investigator has claimed that breaks are produced by MH in cultured mouse fibroblasts (48). Chromosome aberrations did not increase in liver cells of rats fed 1 percent MH in the diet (49). However, the frequency of mitotic cells in adult rat liver is very low. Na–MH does not inhibit mitosis in rats having Walker tumors (17). Results from other mammalian studies, including a dominant lethal test in mice with DEA–MH (50) have not shown increases in chromosomal aberrations.

(2) *Gene Mutations.* Nasrat (51) has reported that maleic hydrazide induced a significant increase ($p<.0003$) in the incidence of sex-linked recessive lethals in *Drosophila melanogaster.* However, there are two possible drawbacks to this study: The number of X chromosomes tested was only 1024, and the raw data are not available to rule out clustering of mutants within a few originally treated males. Clustering of mutations is probably not a problem, because the increase in mutations was noted only in the first brood which samples postmeiotic cells. Only when premeiotic stem cells are affected could multiple mutant sperm arise from a single mutant cell.

There is not much evidence that MH is mutagenic in prokaryotic systems. The *Salmonella typhimurium* histidine reversion test was negative both with (52) and without (53) microsomal activation. Likewise, MH was not mutagenic in *Escherichia coli* (54). On the other hand, in a phage-bearing strain of *Bacillus megatherium* MH doubled both the frequency of mutation to streptomycin resistance and the frequency with which phage entered the lytic cycle (55). MH also induced mutations in chlorophyll loci in barley (56).

(3) *Other tests bearing on mutagenicity.* MH did not increase the frequency of sister chromatid exchanges in the chromosomes of a Chinese hamster cell line (57). It appears that in all but two mutagenicity studies MH was used and not its salts.

(4) *Conclusion.* Several published investigations of MH's mutagenic capacity reviewed above show that MH produces chromosome aberrations in plant and animal test systems. In addition, there is some evidence that MH induces point mutations in bacteria and plants. Accordingly, the working group recommends that a rebuttable presumption be initiated pursuant to § 162.11(a)(3)(ii)(A).

C. REPRODUCTIVE EFFECTS IN MAMMALIAN SPECIES

40 CFR 162.11(a)(3)(ii)(B) provides that "a rebuttable presumption shall arise if a pesticide's ingredient produces any other chronic or delayed toxic effect in test animals at any dosage up to a level, as determined by the Administrator, which is substantially higher than that to which humans can reasonably be anticipated to be exposed, taking into account ample margins of safety."

(1) *Non-mammalian studies.* Although Kang described reduced egg production and increased abnormalities in embryonic development of fresh water limpets treated with MH (58), he used dosages that were greatly in excess of those which might occur in the environment. Also, the development of a gastropod cannot be related to mammalian development in any way (59). Greulach and co-workers (25) saw no dose-related effects on embryonic growth of amphibians.

Robinson's findings (60) indicate that the reproductive system of pea aphids is adversely affected by translocated MH in bean plants. However, the reproductive system of pea aphids is different from that of humans or other mammals. Thus, these findings may not be considered relevant to mammalian reproduction (61).

Fecundity was not reduced in several generations of *Drosophila melanogaster* reared on a diet containing bean plants and potato tubers grown in soil treated with MH (62).

(2) *Mammalian Studies.* Fischnich and co-workers (63) found that fertility was disturbed in rats maintained on a diet that included tubers from potato plants sprayed with DEA–MH. However, no such effect was observed when the diet included potato tubers treated with DEA–MH after harvest. It was concluded in evaluation of this study (61, 64) that the statistically weak data was inconclusive.

When sows were fed tobacco leaf extract to which MH (2 mg/kg of 58 percent of DEA salt) and a mixed insecticide were added, no embryonic or fetotoxic effects were observed (65). Such adverse effects were observed only when sows were fed tobacco juice and/or stake which were grown without application of any chemical, including maleic hydrazide. This study does not address the effects of translocated MH or its metabolites.

A study on reproductive effects resulting from the feeding of the Na and DEA salts of MH to rats was provided to the Agency by a registrant (41). While no significant reproductive effect

[3] The LD_0 for MH injected subcutaneously in mice in 8,800 mg/kg; that for MH injected subcutaneously in rats in 1,300 mg/kg. The LD_{50} for MH administered orally in rats is 3,800 mg/kg (36).

can be noted for rats fed from 0.5 percent to 5 percent Na-MH, significant differences in reproductive effects can be estimated from data provided for rats fed control diets and for rats fed the single tested dose of 0.2 percent DEA-MH. The fertility (pregnancies/matings), viability (pups alive at 4 days/pups born) and the lactation (pups weaned/pups alive at 4 days) are significantly reduced for DEA-MH fed rats in this study. While the average number of pups/litter for DEA-fed rats is less than half the litter size of control rats (4.3 compared to 8.8), the Working Group can not estimate from the study data whether this reduction is significant. The table below amplifies the statement above:

TABLE 4.—*Statistics on reproductive effects due to DEA-MH*

	Control	DEA-MH	Percent
1. Fertility (pregnancies/matings)	40/56	20/41	0.05
2. Viability (pups alive at 4 d/pups born)	280/333	37/73	.001
3. Lactation (pups weaned/pups alive at 4 d)	213/280	21/37	.05

From the data in this table the Working Group concludes that a daily diet containing DEA-MH causes pronounced reproductive effects in rats. Such a diet is equivalent to a daily ingestion of 100 mg/kg of DEA-MH. A comparable diet for a 60 kg man would result in an exposure of 6000 mg/person/day. Based on the consumption figures and tolerances for DEA-MH on onions and potatoes presented earlier in this document, the average daily intake of MH in the DEA-MH formulation is calculated at 5.5 mg/person/day.

The Pesticide Chemical Review Committee (PCRC) is concerned about the proximity of the estimated exposure level (5.5 mg/day) to the observed reproductive-effect level in rats extrapolated to man (6000 mg/day). Their concern is increased by the recognition that the study presents only data on an effect level without using other dosages to establish a dose-response relationship and a no-observable-effect level. If such a study had been performed, the resulting no-observable-effect level would have been lower than the 6000 mg/day dosage which produced significant decreases in three reproductive parameters. Thus, the no-effect and exposure levels would be even closer to one another than what is observed now for the effect and exposure levels. The PCRC concludes that (1) DEA-MH produces significant adverse effects on several reproductive parameters and (2) that an ample margin of safety has not been demonstrated as to the reproductive effects produced by DEA-MH. Therefore, the PCRC recommends that a RPAR be triggered for DEA-MH, but not for K-MH. It has been assumed that K-MH would be similar in its action to that of Na-MH.

(3) Conclusion. Even though the PCRC concludes that DEA-MH (but not Na-MH) meets or exceeds the risk criterion for reproductive effects (40 CFR 162.11(a)(3)(ii)(B), additional data from the registrant is necessary so that the Agency may more precisely estimate the extent of risk caused by the uses of DEA-MH. Consequently, the PCRC and Working Group recommend that affected registrants be required to perform a reproductive study from which dose-response relationships and a no-observable effect level can be determined. In addition, registrants should also be required to submit information on the translocation of DEA-MH to onion bulbs and potato tubers. Further, the PCRC recommends that interested registrants meet with Agency scientists to discuss the adequacy of the protocols developed to meet the above requirements.

IV. *Other Studies.* This section addresses a study which indicates that DEA-MH may produce an adverse effect but for which insufficient evidence exists to issue a rebuttable presumption. The Working Group recommends that the Agency solicit comment on the evidence presented below and encourage the submission of additional studies or relevant information on this effect.

DEA in MH formulations may react with nitrites and nitrates in plants to form N-nitroso compounds (13). In this study N-nitrosodiethanolamine, a metabolite of DEA, in amounts ranging from 145 to 173 ng/g, has been isolated from tobacco treated with DEA-MH. Cigarette tobacco treated with a mixture of the diethanolamine and potassium salts of MH contained 104 ng of N-nitrosodiethanolamine per gram, whereas a flue-cured tobacco treated only with the potassium salt of MH contained 0.1 ng of N-nitrosodiethanolamine per gram (13). Schmeltz and coworkers (13) in their study referenced the work of Druckrey and coworkers (14) which indicated a finding that N-nitrosodiethanolamine is oncogenic. However, this study is not presently available to enable the Working Group to evaluate the work.

The Working Group therefore unanimously agrees that while they do not have enough information to issue an RPAR on N-nitrosodiethanolamine, the Agency should seek more information related to oncogenicity, to other toxic effects of N-nitrosodiethanolamine and to the potential formation of N-nitrosodiethanolamine in other treated plants and in animals exposed to the diethanolamine salt of MH. Should evaluation of this or any other study that comes to our attention provide an adequate basis for an RPAR, a supplemental Position Document and Federal Register notice will be issued.

LIST OF APPENDICES

1. Kutz, F. W. February 15, 1977. Request for human and environmental monitoring data. Memorandum to Robert Brown, OSPR. 2 pp.

2. Levi, E. and A. S. Crafts. 1952. Toxicity of maleic hydrazide in California soils. Hilgardia 21:431. Cited in I. Hoffman and E. V. Parups. 1964. Mode of action of maleic hydrazide in relation to residues in crops and soils. Residue Rev. 7:96–113.

3. Hoffman, I., E. V. Parups, and R. B. Carson. 1962. Analysis for maleic hydrazide. J. Agr. Food. Chem. 10(6):453–455.

4. Fletcher, W. W. 1960. The effect of herbicides on soil micro-organisms. In *Herbicides and the Soil.* E. K. Woodward and G. R. Jagar, eds. Blackwell Scientific Publications. Oxford. pp. 20–52.

5. Smith, A. E., J. W. Zukel, M. Stone, and J. A. Riddle. 1959. Factors affecting the performance of maleic hydrazide. J. Agr. Food Chem. 7(5):341–344.

6. Isenberg. F. R. M. 1954. The effect of maleic hydrazide in plants. Doctoral dissertation. Pennsylvania State University Park, Pa. Cited in I. Hoffman and E. V. Parups. 1964. Mode of action of maleic hydrazide in relation to residues in crops and soils. Residue Rev. 7:96–113.

7. Towers, G. H., M. A. Hutchinson, and W. A. Andreae. 1958. Formation of a glycoside of maleic hydrazide in plants. Nature 181:1535–1536.

8. Shaw, W. C., J. L. Hilton, D. E. Moreland, and L. L. Jansen. 1960. Fate of Herbicides in Plants. In *The Nature and Fate of Chemicals Applied to Soils, Plants and Animals.* USDA. ARS 20–9. pp. 119–133.

Weller, L. E., C. D. Ball, and H. M. Sell. 1957. Studies of maleic hydrazide interactions with thiol compounds. Plant Physiol. 32:146–148.

9. Baker, J. E. 1958. An investigation of the mechanism of action of maleic hydrazide in tobacco and other plants. Doctoral dissertation. North Carolina State College. Raleigh, N.C. Cited in P. R. Datta. 1973. "Mini"—"treetop"—review of maleic hydrazide (MH). EPA. Unpublished.

10. Toth, B. 1975. Synthetic and naturally occurring hydrazines as possible cancer causative agents. Cancer Res. 35:3693–3697.

11. Biswas, P. K., O. Hall, and B. D. Mayberry. 1967. Metabolism of maleic hydrazide in tea, *Camellia sinensis.* Physiol. Plantarum 20:819–824.

12. Rathman, A. April 6, 1977. Memorandum to B. C. Gandhi, Project Manager, OSPR. 4 pp.

13. Schmeltz, I., S. Abidi, and D. Hoffman, 1977. Tumorigenic agents in unburned processed tobacco: N-nitrosodiethanolamine and 1,1-dimethylhydrazine. Cancer Letters 2:125–132.

14. Druckrey, H., R. Preussman, S. Ivankovic, and D. Schmahl. 1967. Organotrope carcinogene Wirkungen bei 65 verschiedenen N-nitroso-Verbindungenan BD-Ratten. Z. Krebsforsch. 69: 103–201. Cited in Schmeltz, I., S. Abidi, and D. Hoffman, *Ibid.*

15. Mays, D. L., G. S. Born, J. E. Christian, and B. J. Liska. 1968. Fate of C^{14}-maleic hydrazide in rats. J. Agr. Food Chem. 16:356–357.

16. El Masri, A. M. 1956. Doctoral dissertation. University of London. Cited in A. Rathman. April 6, 1977. Maleic hydrazide. Memorandum to B. C. Gandhi, Project Manager, OSPR.

17. Barnes, J. M. et al. 1957. The non-toxicity of maleic hydrazide for mammalian tissues. Nature 180:62–64.

18. Comptroller General of the United States. 1974. Questions on the safety of the pesticide maleic hydrazide used in potatoes and other crops have not been answered. Unpublished.

19. Stone, G. M. February 4, 1977. MH residues and MH-30 treatment. Memorandum to B. Gandhi, Project Manager, OSPR. 33 pp.

20. Schmeltz, I., D. Hoffman, and E. L. Wynder. 1974. Toxic and tumorigenic agents in tobacco smoke: Analytical. Methods and

modes of action. In *Trace Substances in Environmental Health—VIII. A Symposium.* D. D. Hemphill, ed. University of Missouri. Columbia, Mo. pp. 281–295.

Liu, Y. Y., I. Schmeltz, and D. Hoffman. 1974. Chemical studies on tobacco and cigarette smoke. Anal. Chem. 46(7):885–889.

21. Harris, T. H. September 4, 1963. Information on pesticide residues on tobacco. Memorandum. 7 pp.

22. Gandhi, B. 1977. MH production and distribution, 1975–76. Memo to files. *Confidential.*

23. Gandhi, G. C. July 5, 1977. Memorandum to files regarding telephone conversation with C. Porter, Commodities Economic Division, Economic Research Service, USDA.

24. Schoene, D. L. and O. L. Hoffman, 1949. Maleic hydrazide, a unique growth regulant. Science 109:588–590.

25. Greulach, V. A., J. McKenzie, and E. M. Stacy. 1951. The effect of maleic hydrazide on the embryonic and larval growth of three amphibians. Biol. Bull. 101:285–288.

26. Dickens, F. and H. E. H. Jones. 1965. Further studies on the carcinogenic action of certain lactones and related substances in the rat and mouse. Brit. J. Cancer 19(2): 1392–1403.

27. Tate, H. D. October 19, 1965. Conference with Drs. Dickens and Jones of Courtauld Institute of Biochemistry, and Chester Beatty Research Institute, Institute of Cancer Research, London, England, October 6th and 7th, 1965. Memorandum to C. D. McCleary. 4 pp.

28. Oser, B. Memorandum for conference on maleic hydrazide, Washington, D.C., October 14, 1965. Food and Drug Research Laboratories, Inc. Maspeth, N.Y. 6 p.

29. Epstein, S. S. and N. Mantel. 1968. Heptocarcinogenicity of the herbicide maleic hydrazide following parenteral administration to infant Swiss Mice. Int. J. Cancer 3: 325–335.

30. Tso, T. C. March 21, 1967. Carcinogenicity of maleic hydrazide. Memorandum to H. A. Rodenhiser, Deputy Administrator, Farm Research, USDA. 1 p.

Rodenhiser, H. A. April 12, 1967. Carcinogenicity of maleic hydrazide. Memorandum to G. W. Irving, Jr. Administrator, ARS, USDA, 1 p.

31. Harris, T. H. November 18, 1969. Memorandum to H. W. Hays, Pesticides Regulation Division, ARS, USDA. 1 p.

32. Hays, H. W. June 22, 1970. Maleic hydrazide. Memorandum to G. W. Irving, Jr., Administrator, ARS, USDA. 1 p.

33. U.S. Department of Health, Education, and Welfare. 1969. Report of the Secretary's Commission on Pesticides and their Relationship to Environmental Health. ("Mrak Report"). pp. 475–579.

34. Innes, J. R. M. et al. 1969. Bioassay of pesticides and industrial chemicals for tumorigenicity in mice: A preliminary note. J. Nat. Cancer Inst. 42:1101–1114.

35. *Registry of Toxic Effects of Chemical Substances. 1975 Edition.* H. E. Christensen and T. Luginbyhl, eds. U.S. Department of Health, Education, and Welfare. Rockville, Md. p. 996.

36. Davido, F. L. February 4, 1977. Pesticide episode information maleic hydrazide (Letter Report No. 12). Report to B. Gandhi, Project Manager, OSPR.

37. Albert, R. E. June 27, 1977. Pre-RPAR evaluation of the carcinogenicity of Maleic Hydrazide. Revised Maleic hydrazide report to B. C. Gandhi, Project Manager, OSPR.

38. Mukhorina, K. V. 1962. Effect of maleic hydrazide, which inhibits the germination of the potato plant, on animal organisms. Gigiena i Toksikol. Novykh Pestisidov. i Klinika Otravlenii Dokl. 20: 156–164.

39. Hunter, B., L. E. Mawdesley-Thomas, and A. N. Worden. 1973. The administration of maleic hydrazide and its diethanolamine salt to rats. Toxicology 1:301–307.

40. Mannell, W. A. and H. C. Grice. 1957. A comparative study of the effects of maleic hydrazide and p-dimethyl-aminoazobenzene on rat liver. Can. J. Biochem. Physiol. 35:1233–1240.

41. Food Research Laboratories. 1958. Toxicological studies of chemical additives. 3 Maleic hydrazide (1,2-dihydropyridazine)3,6 dione. Laboratory No. 62006. Long Island City, N.Y. Unpublished.

42. Mitlehner, A. W. 1971. A summary of toxicity studies with maleic hydrazide. Uniroyal Chemical Division of Uniroyal Inc. Agricultural Chemicals Research and Development. Bethany, Conn. Unpublished.

43. Health Effects Research Laboratory, Office of Research and Development, EPA. May 12, 1977. Toxicological studies of MH. Evaluation report to B. C. Gandhi, Project Manager, OSPR. 1 p.

44. Sandhu S. March 17, 1977. Review of the data to evaluate the mutagenic potential of maleic hydrazide. Evaluation report from Health Effects Research Laboratory, Office of Research and Development, EPA to B. Gandhi, Project Manager, OSPR.

45. Darlington, C. D. and J. MacLeish. 1951. Action of maleic hydrazide on the cell. Nature 167:407–408.

Dubinina, L. G. and N. P. Dubinin. 1968. News in the action of alkylating compounds on chromosome mutations. Soviet Genetics 4:127–153.

Graf, G. E. 1957. Chromosome breakage induced by x-rays, maleic hydrazide and its derivatives. J. Hered. 48:155–159.

Mann, S. K., S. V. Brat, and S. Mhach. 1975. Effect of maleic hydrazide on mitosis. Acta Bot. Indica. 2(1):44–48.

Slotova, J., Z. Karpfel, and D. Kubickova. 1971. The effect of maleic hydrazide and exogenous DNA on chromosomes of *Vicia faba*. Biol. Plantarum (Phaha) 13(5–6): 295–304.

46. Manna, G. K. and B. B. Parida. 1972. Differential maleic hydrazide (MH) sensitivity shown by the spermatocyte chromosomes in five species of grasshopper. Cytologia 37(2):345–353.

47. Manna, G. K., R. K. Das, and P. K. Das. 1976. Chromosome aberrations in mice treated with maleic hydrazide, uracil and guanine. Nucleus 19(1):40–46.

48. McCarthy, R. E. and S. S. Epstein. 1968. Cytochemical and cytogenetic effects of maleic hydrazide on cultured mammalian cells. Life Sciences. 7:1–6.

Shaw, M. W. 1970. Human chromosome damage by chemical agents. Annu. Rev. Med. 21:409–432.

49. Hitachi, M., K. Yamada, and S. Takayama. 1975. Brief communication: Cytologic changes induced in rat liver cells by short-term exposure to chemical substances. J. Nat. Cancer Inst. 54(5):1245–1247.

50. Epstein, S. S., E. Arnold. J. Andrea, W. Bass, and Y. Bishop. 1972. Detection of chemical mutagens by the dominant lethal assay in the mouse. Toxicol. Appl. Pharmacol. 23:288–325.

51. Nasrat, G. E. 1965. Maleic hydrazide, a chemical mutagen in *Drosophila melanogaster*. Nature 207:439.

52. McCann, J., E. Choi, E. Yamasaki, and B. N. Ames. 1975. Detection of carcinogens as mutagens in the *Salmonella*/microsome test: Assay of 300 chemicals. Proc. Nat. Acad. Sci. 72(12):5135–5139.

McCann, J. and B. N. Ames. 1976. Detection of carcinogens as mutagens in the *Salmonella*/microsome test: Assay of 300 chemicals: Discussion Proc. Nat. Acad. Sci. USA 73(3): 950–954.

53. Anderson, K. S., E. G. Leighty, and M. T. Takahashi. 1972. Evaluation of herbicides for possible mutagenic properties. J. Agr. Food Chem. 20(3):649–655.

54. Szybalski, W. 1958. Special microbiological systems. II. Observations on chemical mutagenesis in microorganisms. Annu. N.Y. Sci. 76:475–489.

55. Northrup, J. H. 1963. Studies of the origin of bacterial viruses: VII. The effect of various mutagens (urethane, ethyl urethane, hydrogen peroxide, desoxycholate, maleic hydrazide, butadiene dioxide, triethylene melamine, versene, and acriflavin on the proportion of virus-producing and streptomycin-resistant cells in cultures of *B. megatherium* 20Δ. J. Gen. Physiol. 46:971–980.

56. Kak, S. N. and B. L. Kaul, 1975. Mutagenic activity of hydrazine and its combinations with maleic hydrazide and x-rays in barley. Cytobios. 12:123–128.

Malepsy, S., J. Eberhardt, and M. Maluszynski. 1973. Mutagenic effects of NMH, NEH, and HM in barley. Genetica Polonica 14(1): 47–59.

57. Perry, P. and H. J. Evans, 1975. Cytological detection of mutagen-carcinogen exposure by sister chromatid exchange. Nature 258:121–125.

58. Kang, Y–H. 1972. Reproduction and development of *Ferrissia rivularis* (Say) (Basommatophara: Ancylidae) and the effects of maleic hydrazide on its development and recundity. Doctoral dissertation. Brigham Young University. Provo, Utah p. 234–247.

59. Chernoff, N. March 16, 1977. Letter from Health Effects Research Laboratory, EPA to Robert Brown, OSPR. With attachments. 5 pp.

60. Robinson, A. G. 1960. Effect of maleic hydrazide and other plant growth regulators on the pea aphids, *Acyrthosiphon pisum* (Harris) caged on broad beans, *Vicia Faba L.* Can. Entomol. 92:494–499.

61. Chernoff, N. and R. Kavlock. July 13, 1977. Letter from Health Effects Research Laboratory, EPA to Bipin Gandhi, Project Manager, OSPR. With attachments. 8 pp.

62. Yule, W. M., E. V. Parups, and I. Hoffman. 1966. Toxicology of plant-translocated maleic hydrazide. Lack of effects on insect reproduction. J. Agr. Food Chem. 14(4):407–409.

Yule, W. N., I. Hoffman, and E. V. Parups. 1966. Insect toxicological studies of maleic hydrazide translocated in the potato plant. Bull. Environ. Contamin. Toxicol. 1:251–256.

63. Fischnich, O., C. Patzold, and C. Schiller. 1958. Growth regulating substances on potato crops. Eur. Potato. J. 1(1):25–30.

64. Engler R. June 28, 1974. Review of Article "Growth Regulators in Potato Production * * *" Fischnich, O., C. Patzold, and C. Schiller. Eur. Potato J. 1:25 (1958).

65. Crowe, M. W. and T. W. Swerczrek. 1974. Congenital arthrogryposis in offspring of sows fed tobacco (*Nicotiana tabacum*). Amer. J. Vet. Res. 35(8):1071–1073.

66. K. Diane Courtney. July 25, 1977. Validation report from Toxic Effects Branch, Health Effects Research Laboratory, EPA to Bipin Gandhi, Project Manager, OSPR. 1 p.

67. Svendsgaard, D. July 27, 1977. Statistical report from Statistics and Data Management Office, Health Effects Research Laboratory, EPA to Bipin Gandhi, Project Manager, OSPR. 2 pp.

Hill, R. S. August 10, 1977. Maleic Hydrazide: Mammalian Reproduction Study. Statistical report from Health Effects and Science Policy, Office of Acting Assistant Administrator for Toxic Substances to Bipin Gandhi, Project Manager, OSPR. 1 p.

**** PRODUCT SEARCH LISTING **** PAGE 1

07/27/77 FEDERALLY REGISTERED PRODUCTS CONTAINING MALEIC HYDRAZINE

REGISTRANT *NAME AND ADDRESS*

* 000372 MALLINCKRODT INC
SECOND AND MALLINCKRODT STREETS
ST LOUIS, MO 63160

********** PRODUCT NAME **********

**JJJ43* PO-SAN

REGISTRANT *NAME AND ADDRESS*

* CC0400 UNIROYAL CHEMICAL
DIV UNIROYAL INC
AMITY ROAD
BETHANY CT 06525

********** PRODUCT NAME **********

**00041* MH-30

**00067* SLO-GRO PLANT GROWTH RETARD LABOR SAVER FOR HEDGES, IVY & ROUGH GRA

**JJJ77* GRO-SLO

**00084* ROYAL MH-30 GROW. RETAR.-PREV. TOBACCO SUCKER GROWTH W/SORBATRAN

**00094* ROYAL SLO-GRO GROWTH RETARDANT

**00097* MALEIC HYDRAZIDE TECHNICAL FOR MANUFACTURING USE ONLY

REGISTRANT *NAME AND ADDRESS*

* 000407 IMPERIAL INC
BOX 423
SHENANDOAH IA 51601

********** PRODUCT NAME **********

**CONTINUE REGISTRANT 000407

**0C344* IMPERIAL LIQUID GROWTH RETARDANT

REGISTRANT *NAME AND ADDRESS*

* JJJ456 CHEMICAL FORMULATORS INC
BOX 26
NITRO, WV 25143

********** PRODUCT NAME **********

**0CC97* MALEIC HYDRAZIDE 30

REGISTRANT *NAME AND ADDRESS*

* JJJ551 BAIRD & MCGUIRE INC
SOUTH ST
HOLBROOK MA 02343

********** PRODUCT NAME **********

**00154* BAIRD S MALEIC HYDRAZIDE

REGISTRANT *NAME AND ADDRESS*

* 000554 AGSCO INC
BOX 458
GRAND FORKS ND 58201

********** PRODUCT NAME **********

**00118* AGSCO CHIP 'N STORE POTATO SPROUT INHIBITOR

FEDERALLY REGISTERED PRODUCTS CONTAINING MALEIC HYDRAZINE

REGISTRANT	*NAME AND ADDRESS*
* 000635	E-Z FLO CHEMICAL CO. DIV. OF GROWER SERVICE CORP. P.O. BOX 808 LANSING, MI 48901

********** PRODUCT NAME **********

**00632* E-Z FLO MALEIC HYDRAZIDE (MH-30)

REGISTRANT	*NAME AND ADDRESS*
* 001258	OLIN CHEMICALS OLIN CORPORATION 120 LONG RIDGE ROAD STAMFORD, CT 06904

********** PRODUCT NAME **********

**00865* OLIN MALEIC HYDRAZIDE 2 LBS EMULSIFIABLE

REGISTRANT	*NAME AND ADDRESS*
* 001266	MALTER INTERNATIONAL CORPORATION PO BOX 6055 NEW ORLEANS LA 70114

********** PRODUCT NAME **********

**00121* D-LAY

FEDERALLY REGISTERED PRODUCTS CONTAINING MALEIC HYDRAZINE

REGISTRANT	*NAME AND ADDRESS*
* 001553	MOMAR INC PO BOX 15567 STATION N ATLANTA GA 30325

********** PRODUCT NAME **********

**00097* MOMAR SLOW-UP

REGISTRANT	*NAME AND ADDRESS*
* 001769	NATIONAL CHEMSEARCH DIV USACHEM INC 2727 CHEMSEARCH BLVD IRVING TX 75060

********** PRODUCT NAME **********

**00195* NATIONAL CHEMSEARCH CRO-TARD LIQUID GROWTH RETARDANT FOR GRASS

REGISTRANT	*NAME AND ADDRESS*
* 001926	NAVY BRAND MFG COMPANY 5211 SW AVE ST LOUIS MO 63110

********** PRODUCT NAME **********

**00054* SLOWS-IT (TO RETARD PLANT GROWTH)

**** PRODUCT SEARCH LISTING **** PAGE 5

07/27/77 FEDERALLY REGISTERED PRODUCTS CONTAINING MALEIC HYDRAZINE

REGISTRANT *NAME AND ADDRESS*

* 002125 SCIENCE PRODUCTS COMPANY INC
5831 N TRIPP AVE
CHICAGO, IL 60646

********** PRODUCT NAME **********

**00031* SCIENCE STOP GRASS

**00044* SCIENCE HEDGE TRIM

REGISTRANT *NAME AND ADDRESS*

* 002155 SCHNEIDI.INC.
1429 FAIRMONT AVE N W
ATLANTA GA 30318

********** PRODUCT NAME **********

**00082* BITE MITE CONCENTRATE

REGISTRANT *NAME AND ADDRESS*

* 002169 PATTERSON CHEMICAL COMPANY INC
1400 UNION AVE
KANSAS CITY, MO 64101

********** PRODUCT NAME **********

**00235* PATTERSONS GROWTH CONTROL

**** PRODUCT SEARCH LISTING **** PAGE 6

07/27/77 FEDERALLY REGISTERED PRODUCTS CONTAINING MALEIC HYDRAZINE

REGISTRANT *NAME AND ADDRESS*

* 002270 HUGE COMPANY INC
7625 PAGE AVE
ST. LOUIS, MO 63133

********** PRODUCT NAME **********

**00563* NOGROW LIQUID GROWTH RETARDANT

REGISTRANT *NAME AND ADDRESS*

* 002745 ACETO CHEMICAL COMPANY INC
126-02 NORTHERN BLVD
FLUSHING NY 11368

********** PRODUCT NAME **********

**00044* MALEIC HYDRAZIDE TECHNICAL

**00048* SUCKER CONTROL

**00049* MALEIC HYDRAZIDE SPROUT STOP

**00058* STUNT MAN LIQUID GROWTH RETARDANT

**00124* ACETO LIQUID LAWNMOWER

REGISTRANT *NAME AND ADDRESS*

* 002853 VINELAND CHEM COMPANY
PO BOX 745
VINELAND NJ 08360

********** PRODUCT NAME **********

**00040* MALEIC HYDRAZIDE - 30% PLANT GROWTH REGULATOR

07/27/77 FEDERALLY REGISTERED PRODUCTS CONTAINING MALEIC HYDRAZINE

REGISTRANT *NAME AND ADDRESS*

* 003743 SOUTHERN AGRICULTURAL CHEMICALS INC
PO BOX 527
KINGSTREE SC 29556

********** PRODUCT NAME **********

**00297* MALEIC HYDRAZIDE 30%

REGISTRANT *NAME AND ADDRESS*

* 003770 ECONOMY FEED COMPANY
BOX 237
HOSPERS, IA 51238

********** PRODUCT NAME **********

**00274* SLOW GROW LIQUID GROWTH RETARDANT

**00275* NO SPROUT LIQUID GROWTH RETARDANT

REGISTRANT *NAME AND ADDRESS*

* 004185 SMITH-DOUGLASS DIV.
BORDEN CHEMICAL, BORDEN INC.
5100 VIRGINIA BEACH BLVD.
NORFOLK, VA 23501

********** PRODUCT NAME **********

**00445* SMITH DOUGLASS SUCKER STYMIE

**00487* SUCKER STYMIE PLUS

**00529* 3# MALEIC HYDRAZIDE

07/27/77 FEDERALLY REGISTERED PRODUCTS CONTAINING MALEIC HYDRAZINE

REGISTRANT *NAME AND ADDRESS*

* 004828 ABCO INC
230 INDUSTRY BLVD
NORTH HUNTINGTON, PA 15642

********** PRODUCT NAME **********

**00088* IMPEDE

REGISTRANT *NAME AND ADDRESS*

* 004990 JET-AER CORPORATION
100 SIXTH AVE
PATERSON NJ 07524

********** PRODUCT NAME **********

**CCC19* HEDDY HEDGE CONTROL

REGISTRANT *NAME AND ADDRESS*

* 005481 AMVAC CHEMICAL CORP
4100 EAST WASHINGTON BLVD
LOS ANGELES, CA 90023

********** PRODUCT NAME **********

**00177* ALCC STOP GROW

**** PRODUCT SEARCH LISTING **** PAGE 9

07/27/77 FEDERALLY REGISTERED PRODUCTS CONTAINING MALEIC HYDRAZINE

REGISTRANT *NAME AND ADDRESS*

* 005905 HELENA CHEMICAL CO
CLARK TOWER, 5100 POPLAR AVE, SUITE 2904
MEMPHIS TN 38137

********** PRODUCT NAME **********

**00224* HELENA BRAND 30% MALEIC HYDRAZIDE

**00360* THOR BRAND MALEIC HYDRAZIDE

REGISTRANT *NAME AND ADDRESS*

* 008123 MILLER FRANK & SONS INC
13831 SO EMERALD AVE
CHICAGO IL 60627

********** PRODUCT NAME **********

**00042* MILLERS GROWTH RETARDANT

REGISTRANT *NAME AND ADDRESS*

* 008590 AGWAY INC
CHEMICAL DIV BOX 1333
SYRACUSE NY 13201

********** PRODUCT NAME **********

**00385* AGWAY INHIBITOR 3E

**** PRODUCT SEARCH LISTING **** PAGE 10

07/27/77 FEDERALLY REGISTERED PRODUCTS CONTAINING MALEIC HYDRAZINE

REGISTRANT *NAME AND ADDRESS*

* 009313 ROSE CHEMICAL PROD INC
PO BOX 23375 545 STIMMEL RD
COLUMBUS OH 43223

********** PRODUCT NAME **********

**00021* BRAKE

REGISTRANT *NAME AND ADDRESS*

* 009618 TRANS CHEMIC INDUSTRIES, INC
1 PENN PLAZA
NEW YORK, NY 10001

********** PRODUCT NAME **********

**00009* T.C.I. MALEIC HYDRAZIDE 99

REGISTRANT *NAME AND ADDRESS*

* 009663 CRYSTAL MFG CORP
1525 N POST OAK RD
HOUSTON TX 77055

********** PRODUCT NAME **********

**00004* MALEIC HYDRAZIDE 30%

**00010* SUPREME SUCKER BAN

REGISTRANT	*NAME AND ADDRESS*
* 000775	RIVERSIDE CHEM COMPANY P.O. BOX 171155 855 RIDGE LAKE BLVD MEMPHIS TN 38117

********** PRODUCT NAME **********

**00222* RIVERSIDE D.H. 30% MALEIC HYDRAZIDE

REGISTRANT	*NAME AND ADDRESS*
* 01CC88	ATHEA LABORATORIES INC 4180 N FIRST ST MILWAUKEE WI 53212

********** PRODUCT NAME **********

**00028* LIQUID GROWTH RETARDANT

REGISTRANT	*NAME AND ADDRESS*
* 010155	GLOBE CHEMICALS INC 45 BURBANK DR TOLEDO OH 43607

********** PRODUCT NAME **********

**00012* GLOBE CHECKMATE

REGISTRANT	*NAME AND ADDRESS*
* 010337	FAIRMOUNT CHEMICAL COMPANY INC 117 BLANCHARD ST NEWARK NJ 07105

********** PRODUCT NAME **********

**00001* DE-SPROUT

**00002* DE-CUT TO CONTROL & RETARD PLANT GROWTH IN AREAS DIFFICULT TO MAINTAIN

**00003* SUPER DE SPROUT (FOR THE PREVENTION OF GROWTH OF TOBACCO SUCKERS)

REGISTRANT	*NAME AND ADDRESS*
* 011488	DYNACHEM INDUSTRIES INC 430 13TH ST CARLSTADT NJ 07072

********** PRODUCT NAME **********

**00002* DYNAZINE 300 PREVENTS GROWTH OF TOBACCO SUCKERS

REGISTRANT	*NAME AND ADDRESS*
* 011474	SUNGRO CHEMICALS INC PO BOX 24632 LOS ANGELES CA 90024

********** PRODUCT NAME **********

**00005* REGULATE GROWTH REGULANT

07/27/77 FEDERALLY REGISTERED PRODUCTS CONTAINING MALEIC HYDRAZINE

REGISTRANT *NAME AND ADDRESS*

* 011511 SHALCO CHEMICAL CORPORATION
2421 LEXINGTON AVENUE
TOLEDO OH 43606

********** PRODUCT NAME **********

**00019* SHALCO GROWTH REGULATOR

REGISTRANT *NAME AND ADDRESS*

* 011515 ABC CHEMICAL CORPORATION
17000 W. EIGHT MILE ROAD
SOUTHFIELD, MI 48075

********** PRODUCT NAME **********

**00C50* NC. 405 GROWTH RETARDANT

REGISTRANT *NAME AND ADDRESS*

* 011547 SHARE CORPORATION
7821 N. FAULKNER RD. P.O. BOX 23053
MILWAUKEE, WI. 53223

********** PRODUCT NAME **********

**00CC3* LIQUID GROWTH RETARDANT

07/27/77 FEDERALLY REGISTERED PRODUCTS CONTAINING MALEIC HYDRAZINE

REGISTRANT *NAME AND ADDRESS*

* 011581 OTUSKA CHEMICAL COMPANY, LTD.
C/O WESTVILLE CHEM CO
104 S. MICHIGAN AVENUE RM 400
CHICAGO, IL 60603

********** PRODUCT NAME **********

**00001* TECHNICAL MALEIC HYDRAZIDE FOR MANUFACTURING PURPOSE

REGISTRANT *NAME AND ADDRESS*

* 015713 DREXEL CHEMICAL COMPANY
2602 CORPORATE AVE E STE 1 BOX 31323
MEMPHIS, TN 38132

********** PRODUCT NAME **********

**00001* DREXEL SUPER-P 225 LIQUID GROWTH RETARDANT FOR PREVENTING GROWTH O
**00002* DREXEL MALEIC HYDRAZIDE 30%
**00005* DREXEL SUPER P 15C LIQUID GROWTH RETARDANT
**00014* SUCKER STUFF LIQUID GROWTH RETARDANT
**00015* RETARD LIQUID GROWTH RETARDANT
**00016* SPROUT-STOP LIQUID GROWTH RETARDANT
**00017* MALEIC HYDRAZIDE 2P
**00020* SUPER SUCKER-STUFF LIQUID GROWTH RETARDANT
**00022* SUPER SUCKER-STUFF H.C.
**00025* MALEIC HYDRAZIDE TECHNICAL GRADE
**00026* MALEIC HYDRAZIDE TECHNICAL GRADE

**** PRODUCT SEARCH LISTING **** PAGE 1

07/27/77 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING MALEIC HYDRAZINE

REGISTRANT	*NAME AND ADDRESS*
* 001812	GRIFFIN CORP. P.O. BOX 1847 VALDOSTA, GA 31601

********** PRODUCT NAME **********

**03876* MALEIC HYDRAZIDE 30

REGISTRANT	*NAME AND ADDRESS*
* 001526	NAVY BRAND MFG COMPANY 5111 SW AVE ST LOUIS MO 63110

********** PRODUCT NAME **********

**03506* SLOWS-IT

REGISTRANT	*NAME AND ADDRESS*
* 007587	DRAKE CHEMICAL COMPANY WEST RIVER ROAD PERRYSBURG OH 43551

********** PRODUCT NAME **********

**09123* DRAKE SLO-UP

**** PRODUCT SEARCH LISTING **** PAGE 2

07/27/77 APPLICANTS FOR REGISTRATION OF PRODUCTS CONTAINING MALEIC HYDRAZINE

REGISTRANT	*NAME AND ADDRESS*
* 010337	FAIRMOUNT CHEMICAL COMPANY INC 117 BLANCHARD ST NEWARK NJ 07105

********** PRODUCT NAME **********

**04406* FAIRMOUNT BRAND "FST-7" CONTACT-TOBACCO SUCKER CONTROL

REGISTRANT	*NAME AND ADDRESS*
* 037347	UNI-CHEM CORP. OF FLORIDA BOX 6336 - 2801 N.W. 55TH CT. FT. LAUDERDALE, FL 33310.

********** PRODUCT NAME **********

**03125* GRO-TARD CONCENTRATE

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